

impaired glucose tolerance and higher inflammatory status such as an inflammatory disease, cardiac syndrome X and infection may affect EAT, therefore if provided they would be valuable. In addition, inter-observer and intra-observer variability for EAT measurement are asked, which had already been provided.

We accept that above-mentioned additional factors may have effects on EAT. We checked them and the existing data was provided herein. EAT was correlated to uric acid, glucose and C-reactive protein (CRP) but not creatinine, liver functions and duration of HT in our data. On the other hand, we had performed a multivariate analysis including these related parameters and we had determined that left ventricular mass (LVM) is independently related to uric acid and glucose as well as EAT, but not CRP.

In our opinion, the possible effects of increased epicardial adipose tissue on vasculature and heart an active local paracrine role and passive thermogenic effect or systemic endocrine effects are possible mechanisms for active participation of EAT in this process. We believe that further studies on LVM are needed to clarify more accurately the mechanisms and possible causative cells, cytokines and may be receptors and to confirm the importance of modulating real underlying mechanism to improve clinical outcome.

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LDL cholesterol measurement in terms of CHOLINDEX

LDL kolesterol ölçümünün CHOLINDEX açısından değerlendirilmesi

Dear Editor,

I have read the manuscript of Akpınar et al. (1), which was published in the Anatolian Cardiology Journal on March 2013 entitled 'A new index (CHOLINDEX) in detecting coronary artery risk' with great enthusiasm and interest. Authors have validated a new index, which can be applicable to our daily practice, while taking care of our patients.

As we understand from the manuscript that serum level of LDL cholesterol was measured by enzymatic method utilizing an auto analyzer. However in our daily practice, we all know that when we order 'Lipid profile' for our patients, whether in a university or a government or a special hospital, lipid profile results mostly reported with an indi-

rect formulated measurement, which is Friedewald formula. This formula was validated in 1972 and still inside the market. Besides this formula there are new formulas under investigation and still validating for our daily practice. These new automated LDL measurement formulas are competing with the former Friedewald formula (2, 3).

The gold standard method for LDL-cholesterol measurement is ultra-centrifugation followed by beta-quantitation, which is expensive and inconvenient for routine clinical application (4). More recently, direct methods of LDL cholesterol measurement using specifically designed detergents have been developed, which outperform those based on inhibition with monoclonal antibodies (5, 6). However, these methods are still quite expensive for most laboratories, and thus direct determination of LDL cholesterol is uncommon in most laboratories worldwide.

I suggest to authors that, if they want to validate this new index for our daily practice, they have to adjust their new index according to Friedewald as well as the newly described validated indirect LDL cholesterol measurement formulas. Otherwise because of time and financial shortage of most of the hospitals, this new Cholindex may not find its value for the scientific and cardiologic assessment of coronary artery disease.

As I conclude, after adjustment of the Cholindex to indirect LDL cholesterol measurements (Friedewald, de Cordova CM) we all can be happy with this new coronary artery disease index.

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Author's Reply

Dear Editor,

We have read the letter to the editor with great interest. Firstly, thank you for your gentle compliments and constitutive comments on the paper (1). We think that author detected very important finding in our manuscript.

Even though we suppose all content of low-density lipoprotein-cholesterol (LDL-C) molecules are the same in clinical practice, because of significant differences between the LDL-C measurement methods in the laboratory, there are various different LDL molecules. As is known, when the Friedewald formula was used, LDL-C molecules include intermediate-density lipoprotein (2). In addition, a new formula was defined for measurement of LDL-C (3) recently.

We are planning a new study with greater patient size according to the suggestion of the author, and we plan to use our formula (CHOLINDEX) in all of the old and new LDL-C measurement formulas (Friedewald, de Cordova CM) and ultracentrifugation followed by beta-quantitation methods.

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Does left ventricular function deteriorate in patients with nasal polyposis?

Nazal polipozisli hastalarda sol ventrikül fonksiyonları bozuluyor mu?

Dear Editor,

We read with great interest the recent article entitled "Evaluation of right ventricular functions in patients with nasal polyposis: an observational study" written by Şimşek et al. (1). They aimed to assess the right ventricular functions in patients with nasal polyposis using the strain and strain rate echocardiography. They showed a subclinical deficit of the right ventricular longitudinal functions in patients with nasal polyposis who are considered to have normal right ventricular

functions. We believe that these findings will enlighten further studies about echocardiographic evaluation of patients with nasal polyposis. Thanks to the authors for their valuable contribution.

Nasal polyposis (NP) is a chronic inflammatory disorder of nasal and sinus mucosa. Larger nasal polyps can block nasal passage and may result in hypoxia and hypercapnia. Cardiovascular complications of NP depend on chronic upper airway obstruction. It has been clearly shown that right ventricle function is impaired in various diseases due to chronic hypoxia (2). However, there is little information on the left ventricular (LV) function in patients with chronic hypoxia. Although LV systolic function was preserved, diastolic function was impaired in hypoxia. Ventricular interaction may impair LV diastolic function (3). Obstructive sleep apnea is another cause of chronic hypoxia and can lead to cardiovascular disturbances. Altekin et al. (4) evaluated LV longitudinal functions with two-dimensional strain echocardiography and showed that OSA deteriorates LV systolic function, and the degree of deterioration is proportionate with the disease severity.

The current study (1) assessed the right ventricular function using the strain and strain rate echocardiography but not LV function. We strongly believe that future large-scale prospective studies are needed to examine the LV function in patients with NP. On the other hand, it would be better, if they also evaluated right ventricular function using several parameters including right ventricular index of myocardial performance, tricuspid annular plane systolic excursion, and myocardial acceleration during isovolumic contraction, right ventricular fractional area change. Because these quantitative measurement are simple and reproducible, and they does not require sophisticated equipment or prolonged image analysis.

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